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Lumbar Fusion: A Defensible Option for Discogenic Low Back Pain?

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Chronic low back is one of the most common reasons that patients seek medical attention. There is little agreement on how to treat pain that persists despite an adequate trial of conservative care. When the pain generator can be determined, a logical and targeted treatment plan can be implemented. Diskography can be used to identify an abnormal, painful disk, although the value and safety of this diagnostic tool have been debated. Lumbar fusion for chronic low back pain remains a contentious issue that has been widely debated; however, many studies have inherent flaws that weaken their conclusions. Procedures that do not include interbody fusion ignore the role of the painful disk and might be associated with poorer results. With proper attention to selecting the correct patient, identifying the correct diagnosis, and choosing the correct procedure, good outcomes can be achieved with lumbar fusion for discogenic low back pain.
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Chronic low back pain is the second most common reason for presentation to primary care providers and results in a substantial economic burden in the United States. Established treatment options include primarily nonoperative modalities, including weight loss and activity modification, structured physical therapy programs, including behavioral and cognitive components, anti-inflammatory medications, and targeted corticosteroid injections. Lumbar fusion for patients with low back pain has traditionally been reserved for patients with associated pathology, including lumbar stenosis, spondylolisthesis, and radiculopathy.

Lumbar fusion has been advocated by some as a viable treatment option for discogenic low back pain in patients who have failed extensive therapy and conservative treatment. The rationale for such treatment is that lumbar fusion results in elimination of the principal pain generator, the painful disk. However, fusion in this setting has been widely debated in the literature and remains controversial.

The purpose of this review is to evaluate existing literature and current practice trends to determine the extent to which lumbar fusion for the treatment of chronic low back pain can be validated as a legitimate treatment option.

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Low Back Pain: Etiology and Diagnosis

Successful treatment of chronic low back pain is not likely to occur without first establishing an accurate diagnosis. To do this, it is imperative to identify as precisely as possible the anatomical location of the pain generator. The many causes of low back pain include muscular or ligamentous strains, degenerative disk disease, facet pathology, diffuse spondylosis, osteoporotic compression fractures, infection, and neoplasms. Intra-abdominal problems such as ulcers and aortic aneurysms require a higher index of suspicion and prompt treatment. Many of the musculoskeletal causes of low back pain can be accurately diagnosed with a good history and physical examination with or without plain radiographs. Occasionally more advanced imaging with computed tomography (CT) and/or magnetic resonance imaging (MRI) might be necessary to establish a diagnosis or guide further evaluation.

Although many degenerative conditions in the lumbar spine undoubtedly overlap, such as the simultaneous presence of disk degeneration and facet arthritis, an appreciation of the predominant pain generator might help in determining surgical approaches. Discogenic low back pain is defined as pain emanating solely from a diseased lumbar disk and associated structures. Pure discogenic pain assumes the absence of radicular pain as well as pain from other structures in the lumbar spine, including the facet joints, ligaments, and muscles.

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The pathogenesis of disk degeneration has been well-described. Changes that occur begin as early as the second decade and are very similar to those associated with disk aging. The normal disk is avascular and obtains nutrition through passive and active transport of molecules through the disk substrate. As vascular penetration across the endplate diminishes, cell death in the disk occurs, resulting in reduced extracellular matrix production and a change in the relative composition of matrix proteoglycans. Matrix metalloproteinases, such as stromelysin and collagenases, which are responsible for the normal turnover of disk matrix, begin to degrade matrix components at an increased rate. This in turn leads to reduced ability of the disk to attract and maintain water concentration, leading to dehydration of the disk and loss of structural capabilities.

Annular strain increases, and tears of the annulus also occur. At the periphery, vascular ingrowth and penetration of nerve fibers into the outer nucleus occur, in association with the production of nerve growth factor.[1] This might sensitize the disk and make it more susceptible to pain from mechanical loading. In addition, interleukin-1 (IL-1) has been identified in degenerative disks, which is responsible for the generation of various pain mediators.[2] End-stage disk degeneration is accompanied by collapse of disk height and onset of arthritic changes, including endplate sclerosis and peripheral osteophyte formation. Disk space collapse leads ultimately to increased strain and abnormal loading of the facet joints, resulting in facet arthrosis, as well as instability and spondylolisthesis. Disk degeneration is believed by some to be the anatomical starting point for lumbar spine degeneration.

When radiographic signs of discogenic pain are present, such as disk space narrowing and endplate sclerosis, the diagnosis is suggested. However, in some cases plain radiographic changes might not be present, and evidence of disk degeneration might only be seen on MRI, the so-called dark disk. Degenerative disks have diminished water content and are therefore darker on T2-weighted images than their normal counterparts.

However, degenerative disk disease can be seen radiographically in asymptomatic patients,[3] and routine clinical evaluations are unable to establish a definitive diagnosis in up to 85% of cases.[4] When investigating a group of subjects with no back pain but who were age, gender, and occupation matched to low back pain patients, 76% of asymptomatic subjects had at least 1 abnormal disk on MRI.[5] Therefore, disk changes alone on MRI in a patient with low back pain are not a reliable indicator of the source of pain.

Diskography

If the pain generator is still uncertain, provocative tests might be of benefit, including targeted blocks (nerve root, facet, sacroiliac joint) and diskography. Diskography is a study to determine the internal anatomy of the disk that as of yet MRI cannot do routinely. Contrast (typically 2-3 mL) is injected centrally into the disk, and the pattern of dye permeation is noted. The dye is slowly injected until a firm end point is felt.

Table 1 Guidelines for the Use of Diskography

1. **Further evaluation of demonstrably abnormal disks to help assess the extent of abnormality or correlation of the abnormality with the clinical symptoms. Such might include recurrent pain from a previously operated disk and lateral disk herniation.**
2. **Patients with persistent, severe symptoms in whom other diagnostic tests have failed to reveal clear confirmation of a suspected disk as the source of pain.**
3. **Assessment of patients who have failed to respond to surgical procedures to determine whether there is painful pseudarthrosis or a symptomatic disk in a posteriorly fused segment, or to evaluate possible recurrent disk herniation.**
4. **Assessment of disks before fusion to determine whether the disks within the proposed fusion segment are symptomatic and to determine whether disks adjacent to this segment are normal.**
5. **Assessment of minimally invasive surgical candidates to confirm a contained disk herniation or to investigate dye distribution pattern before chemonucleolysis or other intradiscal procedures.**

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If there is a response, the patient is questioned as to the location of the pain to determine clinical relevance. After the dye injection, an injection of a local anesthetic can be performed, which should relieve the patient's pain. A control (normal) disk is often injected as well. For the test to be positive, the disk should appear abnormal, and the patient's typical pain should occur as a result of the injection. Ideally the pain should subside with injection of local anesthetic, and injection of the control disk should be negative. Recommendations for the use of diskography are listed in Table 1.[6]

Diskography has been used for the last several decades. In 1948 Lindblom[7] first described disk puncture as a diagnostic test for low back pain. Cloward and Buzaid[8] later advanced the idea and published on the indications and technique of lumbar diskography. Diskography was later championed by Fernstrom[9] and then Crock,[10] who was one of the first proponents of the disk as an occult source of low back pain. There are several benefits of diskography. It provides detailed information about the structure of the disk that cannot necessarily be discerned on MRI. The pain provocation component has been shown to be related to surgical outcome.[11-16] Finally, one can avoid operating on a dark disk on MRI that is not a source of pain. Diskography has been shown to be more sensitive and specific than MRI in the evaluation of discogenic pain.[17,18]

Despite these benefits, the value and even safety of diskography have been debated over the years. Criticisms of diskography include a high false-positive rate, increased risk of complications, and the potential for iatrogenic disk injury and degeneration. Holt[19] was the first to question the value of diskography after he published an article detailing his results with a study population of asymptomatic inmates. In that widely cited study, pain was elicited in all patients after disk injection, and dye leakage was seen in 93% of cases. This